We claim:

1. A hGR 1Ap/e gene of the human glucocorticoid receptor promoter 1A and exon 1A comprising at least 2056 bases of SEQ ID NO: 1.

2. A hGR 1Ap/e gene as in Claim 1, wherein the promoter region comprises the region from -1075 to -1of SEQ ID NO: 1 as numbered in Figure 1.

3. A hGR 1Ap/e gene as in Claim 1, wherein the exon region comprises the region from +1 to +981 of SEQ ID NO: 1 as numbered in Figure 1.

4. A human glucocorticoid receptor exon 1A region as in Claim 3, wherein transcription of the exon region results in a mRNA transcript.

5. A mRNA transcript of human glucocorticoid receptor exon 1A region as in Claim 4, wherein the transcript results from transcription of the region +1 to +212 of SEQ ID NO: 1 as numbered in Figure 1.

6. A mRNA transcript of human glucocorticoid receptor exon 1A region as in claim 4, wherein the transcript results from transcription of the region +1 to +308 of SEQ ID NO: 1 as numbered in Figure 1.

7. A mRNA transcript of human glucocorticoid receptor exon 1A region as in claim 4, wherein the transcript results from transcription of the region +1 to +981 of SEQ ID NO: 1 as numbered in Figure 1.

8. A method to detect the presence of cancerous lymphocytes in a human, comprising assaying for the expression of the mRNA transcript as in claim 7 in human lymphocytes by using primers chosen from the sequence from +308 to +981 of SEQ ID NO: 1 as numbered in Figure 1.

9. The method of claim 8, wherein the cancerous lymphocytes are T-cell acute lymphoblastic leukemia cells.

10. A method to determine the responsiveness of a patient with cancerous lymphocytes to future treatment with glucocorticoids, comprising isolating lymphoctes from the patient, treating the isolated lymphocytes with glucocorticoid, and assaying for the expression of mRNA transcripts as given in Claim 7 in the treated lymphocytes using primers chosen from the sequence from +308 to +981 of SEQ ID NO: 1 as numbered in Figure 1.

11. A method to increase the expression of mRNA as in claim 7, comprising adding an exogenous substance that causes an increased concentration of interferon regulatory factor, wherein the interferon regulatory factor binds to the DNA sequence somewhere between +102 and +125 in SEQ ID. NO. 1 as numbered in Figure 1.

12. A method as in claim 11, wherein the exogenous substance is interferon.

13. A method to increase the expression of mRNA transcript as in Claim 7 to treat a patient with T-cell acute lymphoblastic leukemia cells, comprising administering to the patient an enhancing amount of exogenous interferon and exogenous glucocorticoid.

14. A method to increase the expression of mRNA transcript as in Claim 7 to treat a patient with T-cell acute lymphoblastic leukemia cells, comprising administering to the patient an enhancing amount of an exogenous demethylating agent to reactivate the human glucocorticoid promoter and exon 1A activity.

15.. The method of claim 14, wherein the demethylating agent is 5-azacytidine.

16. A hGR 1Ap/e promoter-heterologous gene construct comprising all or a portion of SEQ ID NO:1 and a heterologous gene, wherein expression of the heterologous gene of the construct is under transcriptional control of the hGR 1Ap/e promoter.

17. The method of claim 16, wherein the heterologous gene codes for a toxin.

18. A method to kill targeted cells by administering an exogenous dose of glucocorticoid, comprising transforming targeted cells by introducing into said cells the gene construct of claim 17.

19. A method to convert glucocorticoid-resistant lymphoblasts to glucocorticoid-sensitive lymphoblasts, comprising introducing all or a functional portion of SEQ ID NO: 1 into the hormone-resistant lymphoblasts.

20. An antisense transgene comprising all or a functional portion of the promoter region of SEQ ID NO: 1 linked to a fragment of the exon region of SEQ ID NO:1 in the antisense orientation.

21. A method to inhibit hGR1A GR mRNA from being up-regulated in cells, comprising introducing into said cells the antisense transgene of Claim 20.

22. A method to prevent neuronal apoptosis caused by excessive glucocorticoid secretion, comprising introducing into said neuronal cells the antisense transgene of Claim 20.